Journal of Forensic and Legal Medicine 16 (2009) 472–474

Contents lists available at ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/jflm

Case Report

A rare case of myocardial infarction due to parathion poisoning

Yajnesh Kidiyoor MBBS (Post Graduate Student)[a], Vinod C. Nayak MD (Assistant Professor)[b], Vasudha Devi MD (Assistant Professor)[c,*], Shankar M. Bakkannavar MD (Assistant Professor)[b], G. Pradeep Kumar MD (Professor & Head)[b], Ritesh G. Menezes MD, Diplomate NB (Assistant Professor)[d]

[a] Department of General Medicine, A. J. Institute of Medical Sciences, Mangalore, India
[b] Department of Forensic Medicine and Toxicology, Kasturba Medical College, Manipal, India
[c] Department of Pharmacology, Melaka Manipal Medical College, Manipal, India
[d] Department of Forensic Medicine and Toxicology, Kasturba Medical College, Mangalore, India

ARTICLE INFO

Article history:
Received 22 January 2009
Accepted 20 May 2009
Available online 21 June 2009

Keywords:
Organophosphate
Parathion
Poisoning
Myocardial infarction

ABSTRACT

Organophosphorus poisoning occurs very commonly in southern India where farmers form a significant proportion of the population who commonly use organophosphorus compounds like parathion as insecticides. Awareness of the complications caused by parathion poisoning is essential for proper monitoring and treatment. We report a case of suicidal parathion poisoning in a farmer who was treated with atropine and oximes. He subsequently developed myocardial infarction, to the treatment of which, he showed no response and succumbed. In conclusion, myocardial infarction is a rare complication of parathion poisoning. Awareness of this complication of parathion poisoning, proper monitoring and treatment could prevent fatalities.

1. Introduction

Organophosphorus poisoning occurs very commonly in southern India where farmers form a significant proportion of the population who commonly use organophosphorus compounds like parathion as insecticides. Thus, due to the easy accessibility of these compounds a large number of suicidal cases are encountered in this region.[1] Myocardial infarction is one of the complications of organophosphorous poisoning and has been rarely reported.[2–4] Here, we report a fatal case of myocardial infarction due to parathion poisoning. Awareness of this complication following parathion poisoning is essential for proper monitoring and treatment.

2. Case report

A 51-year-old man, a farmer by occupation, was brought to the hospital after he had consumed an unknown quantity of parathion, an organophosphorus compound. He was a known diabetic on oral hypoglycemics for the last 6 years. Otherwise, his medical history did not reveal any significant disease or past hospitalization. On admission, he was drowsy, arousable but did not follow commands. Gastric lavage was performed and chemical analysis of the gastric lavage material showed the presence of parathion. The plasma cholinesterase level showed a drastic reduction (99 IU/l). He was treated with atropine and pralidoxime. He showed signs of improvement, but suddenly his blood pressure dropped on the seventh day of parathion ingestion. A bedside echocardiography was done which suggested the presence of myocardial ischemia. Blood investigations showed a troponin level of 1.0 ng/ml, a CPK level of 137 IU/l and a CK-MB level of 2970 IU/l which suggested severe myocardial infarction. He was treated for the same, but showed no response and succumbed. At autopsy, left anterior descending coronary artery showed 20% obstruction due to atherosclerosis. The histopathological examination of the heart confirmed the presence of a recent myocardial infarction of the anterolateral wall of the left ventricle (Figs. 1 and 2). There were no other significant autopsy findings.

3. Discussion

Organophosphorus poisoning occurs very frequently in southern India as it is commonly employed by farmers as a spray insecticide and this easy availability makes it a preferred suicidal poison. Accidental cases are also reported. Clinical features include salivation, lacrimation, emesis, gastrointestinal cramping, bronchorrhoea, muscle fasciculations, cramps, depression of respiratory centre etc.[5] These are mainly caused due to the accumulation of acetylcholine (Ach) as organophosphorus is an anticholinesterase.

* Corresponding author. Tel.: +91 9945671112; fax: +91 820 2571905.
E-mail addresses: vasudha_manipal@yahoo.co.in, vasudha.devi@manipal.edu (V. Devi).

1752-928X/$ - see front matter © 2009 Elsevier Ltd and Faculty of Forensic and Legal Medicine. All rights reserved.
doi:10.1016/j.jflm.2009.05.003

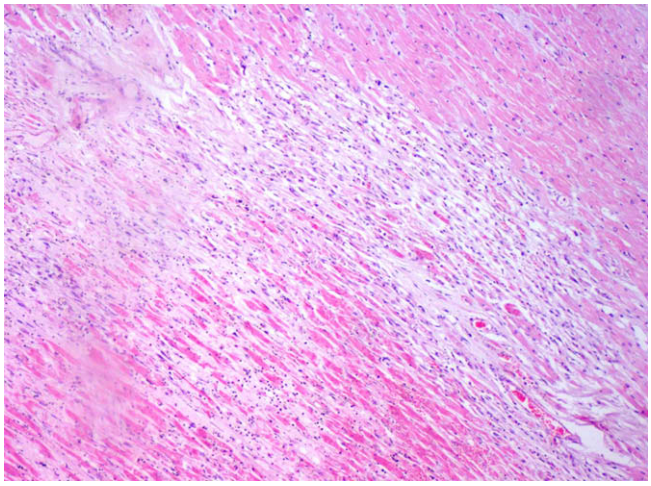

Fig. 1. Acute myocardial infarction (Heamotoxylin and Eosin, 20X). Section shows an area of acute myocardial infarction with myocyte fragmentation, cytoplasmic hypereosinophilia and beginning neutrophilic infiltration.

Coronary spasm plays an important role in the pathogenesis of myocardial infarction. Acetylcholine, generally is a vasodilator acting by releasing endothelial-derived relaxing factor (EDRF).[6] In patients with atheromatous coronary artery Ach causes coronary vasoconstriction even at low doses indicating endothelial dysfunction.[7] The endothelial dysfunction in atherosclerosis also results in an increased sensitivity to the constrictor effects of catecholamines.[8] Furthermore, Ach causes dilatation of angiographically normal coronary arteries and constriction of angiographically normal segments in patients with coronary artery disease suggesting that these segments are functionally abnormal.[9] In addition, the likelihood of Ach-induced constriction of angiographically smooth epicardial vessels is proportional to the number of risk factors for coronary heart disease indicating that coronary risk factors are associated with loss of endothelium-dependent vasodilatation.[10] Furthermore, recently Knezl et al. showed that in streptozotocin-induced experimental diabetic rats, Ach could induce severe dysarrhythmias and Ach induced coronary vasoconstriction last even after washing out the Ach.[11] Newman et al. studied the effect of Ach in patients without coronary artery disease. They showed that Ach concentration of $>10^{-3}$ M causes intense constriction of distal normal epicardial vessels and, in some cases, anginal pain and objective signs of ischemia. Whereas, lower concentration up to 10^{-4} M causes progressive dilatation of left anterior descending artery, but constriction of the distal segments and tertiary branches.[12] It is now well established that the pressor response to Ach is due to the stimulation of M_3 muscarinic receptor in coronary vessels.[13]

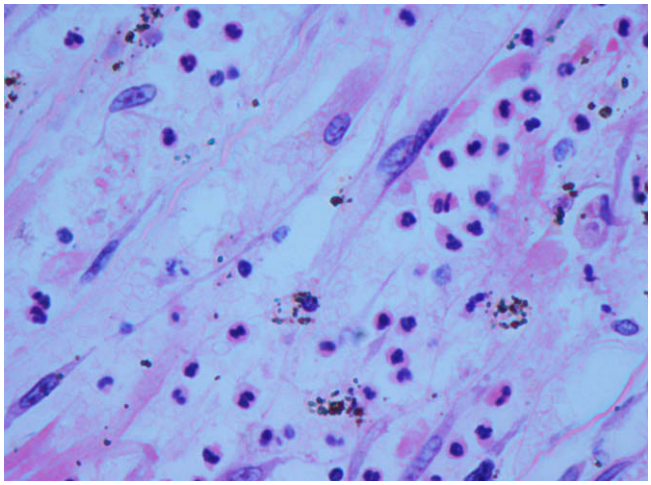

Fig. 2. Acute myocardial infarction (Heamotoxylin and Eosin, 40X). Section shows necrotic myocytes with stromal edema and beginning neutrophilic infiltration.

Mycocardial infarction following organophosphorous poisoning is rarely reported. Lionte et al. have reported a case of a 57-year-old woman who died of myocardial infarction following organophosphorus consumption.[2] Saadeh et al. have studied 46 cases of carbamate and organophosphorus poisoning out of which five showed raised cardiac enzymes.[14] Out of 168 cases of organophosphorus poisoning reported by Kiss and Fazekas, five showed myocardial infarction.[3] Diffuse myocardial infarction was found at autopsy in two cases of malathion poisoning reported by Chharba et al.[4]

Our patient was a farmer who consumed parathion, an organophosphorus insecticide and was treated with atropine and pralidoxime. He showed signs of recovery, but developed myocardial infarction and succumbed. He was a previously healthy individual with no abnormal cardiac history. On autopsy he had non-critical (<50%) diffuse obstruction of left anterior descending coronary artery. The parathion induced increase in Ach concentration might have caused intense spasm of affected coronary artery causing infarction of anterolateral wall of the left ventricle. In addition, presence of coronary risk factors such as uncontrolled diabetes, male gender and >45 years of age might have caused endothelial dysfunction in this patient leading the coronary segments to constrict in response to Ach. Myocardial infarction also may be due to high concentration of Ach induced intense vasospasm of normal distal coronary arteries.

To conclude, we herein report a case of myocardial infarction following ingestion of parathion, which is a very rarely reported complication occurring in organophosphorus poisoning cases. Since there is a strong overall correlation between risk factors and a constrictor response to Ach, carefully eliciting history of coronary risk factors can predict the occurrence of myocardial infarction in patients with parathion poisoning. In addition, early monitoring and treatment could help prevent fatalities.

Conflict of Interest

None declared.

Funding

None declared.

Ethical Approval

None declared.

References

1. Kanchan T, Menezes RG. Suicidal poisoning in Southern India: gender differences. *J Forensic Leg Med* 2008;**15**:7–14.
2. Lionte C, Sorodoc L, Petriş O, Sorodoc V. Electrocardiographic changes in acute organophosphate poisoning. *Rev Med Chir Soc Med Nat Iasi* 2007;**111**(Suppl.):906–11.
3. Kiss Z, Fazekas T. Arrhythmias in organophosphate poisonings. *Acta Cardiol* 1979;**34**:303–23.
4. Chharba ML, Sepaha GC, Jain SR, Bhagwat RR, Khandekar JD. ECG and necropsy changes in organophosphorus compound (malathion) poisoning. *Indian J Med Sci* 1970;**24**:424–9.
5. Cherian MA, Roshini C, Peter JV, Cherian AM. Oximes in organophosphorus poisoning. *Indian J Crit Care Med* 2005;**9**:155–63.
6. Furchgott RF, Zawadzki JV. The obligatory role of the endothelial cells in the relaxation of arterial smooth muscle by acetylcholine. *Nature* 1980;**288**:373–6.
7. Ludmer PL, Selwyn AP, Shook TL, et al. Paradoxical vasoconstriction induced by acetylcholine in atherosclerotic coronary arteries. *New Engl J Med* 1986;**315**:1046–51.

8. Vita JA, Treasure CB, Yeung AC, et al. Patients with evidence of coronary endothelial dysfunction as assessed by acetylcholine infusion demonstrate marked increase in sensitivity to constrictor effects of catecholamines. *Circulation* 1992;**85**:1390–7.
9. Tousoulis D, Davies G, Lefroy DC, Haider AW, Crake T. Variable coronary vasomotor responses to acetylcholine in patients with normal coronary arteriograms: evidence for localised endothelial dysfunction. *Heart* 1996;**75**:261–6.
10. Vita JA, Treasure CB, Nabel EG, et al. Coronary vasomotor response to acetylcholine relates to risk factors for coronary artery disease. *Circulation* 1990;**81**:491–7.
11. Knezl V, Kysel'ová Z, Zúrová-Nedelcevová J, Navarová J, Tribulová N, Drímal J. Effect of acetylcholine and ischaemia/reperfusion injury on the heart of rats with STZ-induced experimental diabetes. *Neuro Endocrinol Lett* 2006;**27**:144–7.
12. Newman CM, Maseri A, Hackett D, El-Tamimi HM, Davies GJ. Response of angiographically normal and atherosclerotic left anterior descending coronary arteries to acetylcholine. *Am J Cardiol* 1990;**66**:1070–6.
13. Hoover DB, Neely DA. Coronary vasoconstrictor response to acetylcholine in isolated perfused rat heart is mediated by M3 muscarinic receptors. *Life Sci* 1995;**56**(1):1041.
14. Saadeh AM, Farrakhan M, Ali-Ali MK. Cardiac manifestations of acute carbamate and organophosphorus poisoning. *Heart* 1997;**77**:461–4.